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Arch Biochem Biophys. 1994 Nov 15;315(1):111-8.
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1: Proc Natl Acad Sci U S A. 1992 Aug 15;89(16):7384-8.

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# Human cyclooxygenase-2 cDNA.

**Hla T, Neilson K.**

Department of Molecular Biology, Holland Laboratory, American Red Cross, Rockville MD 20855.

Cyclooxygenase (Cox), also known as prostaglandin (PG) H synthase (EC 1.14.99.1), catalyzes the rate-limiting step in the formation of inflammatory PGs. A major regulator step in PG biosynthesis is at the level of Cox: growth factors, cytokines, and tumor promoters induce Cox activity. We have cloned the second form of the Cox gene (Cox-2 from human umbilical vein endothelial cells (HUVEC). The cDNA encodes a polypeptid of 604 amino acids that is 61% identical to the previously isolated human Cox-1 polypeptide. In vitro translation of the human (h)Cox-2 transcript in rabbit reticulocyte lysates resulted in the synthesis of a 70-kDa protein that is immunoprecipitated by antiserum to ovine Cox. Expression of the hCox-2 open reading frame in Cos-7 monkey kidney cells results in the elaboration of cyclooxygenase activity. hCox-2 cDNA hybridi to a 4.5-kilobase mRNA species in HUVEC, whereas the hCox-1 cDNA hybridizes to 3- and 5.3-kilobase species. Both Cox-1 and Cox-2 mRNAs are expressed in HUVEC, vascular smooth muscle cells, monocytes, and fibroblasts. Cox-2 mRNA was preferentia induced by phorbol 12-myristate 13-acetate and lipopolysaccharide in human endothelia cells and monocytes. Together, these data demonstrate that the Cox enzyme is encoded b at least two genes that are expressed and differentially regulated in a variety of cell types High-level induction of the hCox-2 transcript in mesenchymal-derived inflammatory cel suggests a role in inflammatory conditions.

PMID: 1380156 [PubMed - indexed for MEDLINE]

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ACCESSION NUMBER: 1994-46604 DRUGU B P E
TITLE: **Characterization** of the mechanism of inhibition of **human cyclooxygenase-2** by anti-inflammatory drugs.
AUTHOR: Ouellet M; Percival M D
CORPORATE SOURCE: Merck-Frosst
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AB The kinetic mechanism of inhibition of the **purified** form of inducible cyclooxygenase (hCox-2) by several classical NSAIDs and a selective Cox-2 inhibitor was determined. Flurbiprofen, meclofenamate and indometacin were all time-dependent inhibitors of hCox-2. None of the 3 inhibitors had a high degree of selectivity, when compared with hCox-1. NS-398 also had time-dependent inhibition of hCox-2, but was a time-independent inhibitor of hCox-1. The difference in the mechanism of inhibition was reflected in the high degree of selectivity observed for hCox-2 over hCox-1. Results demonstrate that the mechanism of inhibition of hCox-2 by classical NSAIDs is similar to that identified for ovine Cox-1. In addition, it shows the nature of time-dependency of inhibition of hCox-1 and hCox-2 greatly determines the degree of selectivity for 1 isozyme over the other. (conference abstract).